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VASCULAR DISEASE

ANGIOTENSINOGEN M235T GENE VARIANTS AFFECT ENDOTHELIAL FUNCTION AND ARTERIAL STIFFNESS IN PATIENTS WITH ESSENTIAL HYPERTENSION

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Background: A common variant of the angiotensinogen (AGT) gene, the M235T, may predict elevated levels of circulating AGT, and may influence the risk of cardiovascular disease. Therefore, we sought to investigate the impact of this polymorphism on the prevalence of essential hypertension (EH) and on vascular properties, such as arterial stiffness and endothelial function.

Methods: Our population consisted of 87 consecutive newly diagnosed, untreated essential hypertensives stage I-II (aged 54±14 years, 47% female), without any history of cardiovascular disease or any other evident comorbidity, and 45 controls. The gene mutation frequency of AGT M235T was determined using polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) technique. Brachial artery flow-mediated dilatation (FMD) has been used to assess endothelial dysfunction. Aortic stiffness was evaluated, on the basis of carotid to femoral pulse wave velocity (c-f PWV) by means of a computerized method (Complior SP).

Results: The M235T AGT genotype distribution in patients with EH (TT = 20.6%, MT = 45.9%, MM = 33.5%) did not differ from genotype distribution in controls (TT = 17.7%, MT = 44.0%, MM = 38.3%), and the TT genotype was not associated with EH (OR 1.1; 95% CI 0.7-1.7; P = 0.6). Interestingly, there was significant difference of FMD between MM and TT genotypes (4.4±2.1 vs 2.5±1.1, $p<0.05$) in hypertensive patients. Similarly, 235T homozygosity was independently associated with decreased levels of FMD compared with MM genotype in healthy individuals, although this difference did not reached statistical significance (3.2±1.2 vs 7.8±3.0, p=NS). In addition, despite the fact that patients homozygous for the T allele had increased arterial stiffness, independently of blood pressure, compared with patients homozygous for the M allele, this difference was not significant (7.7±1.7 vs 6.7±1.3, p=NS).

Conclusions: These data suggest that the AGT TT genotype could be a genetic marker for endothelial dysfunction and arterial stiffness in patients with (never-treated) hypertension. These findings indicate an important mechanism by which AGT II genotype affects vascular function in essential hypertension.